Separating People from Pollution

Individual and Community Interventions to Mitigate Health Effects of Air Pollutants

Efforts to minimize people's exposure to air pollution historically have focused on curbing emissions from tailpipes and smokestacks. But increases in vehicle-kilometers traveled—that is, more cars spending more time on the road—have tempered that effect. Moreover, residential areas, hospitals, and schools often are built adjacent to main traffic arteries, where emissions are highest. An international group of public health researchers now says it's time to start separating people from sources of air pollution as a means of protecting public health [***EHP* 119(1):29–36; Giles et al.**].

Time of day and location affect air pollution exposure during exercise.

Air pollution can cause myriad cardiovascular and respiratory problems including asthma, bronchitis, and heart disease. Outdoor air pollutants can easily migrate indoors, and most exposure to ambient air pollution occurs inside buildings. Recent research indicates that people living near congested highways face a greater risk of such diseases and that moving to a less-polluted neighborhood lowers their risk.

The authors describe "promising and largely unexplored" approaches to reducing the health impact of air pollution through interventions targeted at communities and at individuals. They base their recommendations on published studies and discussions from a 2009 workshop on this topic held in Vancouver, Canada.

The authors argue that cities can improve residents' health by considering air quality during land-use planning. For example, creating high-density, mixed-use areas would enable more people to walk or bicycle to work, school, and shops, thereby reducing emissions and encouraging more exercise; ideally, safe pedestrian and cycling greenways would be located away from traffic. For longer-distance travel, the authors suggest low-emission public transit. And in areas where wood burning is an important heating method, woodstove exchange programs can help residents acquire cleaner-burning stoves affordably.

Risk factors for heart disease include a sedentary lifestyle, obesity, and a high-sodium diet. Therefore, the authors posit that another approach to reducing a person's risk of being affected by air pollution is to minimize one's overall risk of heart disease. This could involve interventions that encourage people to eat a diet rich in omega-3 fatty acids and antioxidants and to get regular exercise. However, because pollution levels vary even within cities, exercise should be planned to minimize exposure. Variations occur by season, with ozone being higher in the summer and particulates from woodstoves higher in the winter, for example. Traffic-related pollutants also spike during rush hour and are higher in heavily traveled areas.

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Estrogens from the Outside In

Alkylphenols, BPA Disrupt ERK Signaling *in Vitro*

The body produces estrogens—including estrone (E_1), estradiol (E_2), and estriol (E_3)—that direct reproductive system processes and contribute to the normal function of tissues including the brain, bone, and cardiovascular system. Certain xenoestrogens (estrogenic compounds introduced from outside the body) are suspected of disrupting these activities. In a new study, xenoestrogenic alkylphenols and bisphenol A (BPA) interfered with normal estrogenic signaling *in vitro*, which suggests they could disrupt normal physiologic function at critical life stages [***EHP* 119(1):104–112; Jeng and Watson**].

Different estrogen receptors control different functions: receptors in the cell nucleus direct gene transcription, whereas receptors in the cell membrane direct signaling pathways via extracellular signal–regulated kinases (ERKs). ERK-controlled pathways respond to many biochemical stimuli and integrate these signals to direct a cell toward division, differentiation, death, or malignant transformation. The structurally related alkylphenols and BPA interact weakly with nuclear estrogen receptors, but they can have pronounced effects on signaling pathways mediated by estrogen receptors in the cell membrane.

In the current study, a rat pituitary cancer cell line was used to study the effect of alkylphenols and BPA on ERK1 and ERK2 activation (measured as phosphorylation), both alone and in combination with each physiologic estrogen. After treatment with each physiologic and environmental estrogen, the researchers measured time-dependent surges in ERK activation. In most cases, E_1 and E_2 prompted early, intermediate, and late surges in ERK activation at 5, 10–30, and > 30 min, respectively; alkylphenols and E_3 typically triggered early and late surges. Interestingly, a very low concentration of BPA (10^{-14} M) yielded a similar two-peak response, but a higher concentration (1 nM) induced a three-peak response like that of E_1 and E_2. Both BPA concentrations were typical of environmental exposures and, along with ineffective midrange doses, also illustrated the nonmonotonic dose–response relationship characteristic of many estrogenic compounds.

When physiologic estrogens and xenoestrogens were combined, the response pattern generally shifted to a single major peak at an intermediate time. Xenoestrogens that caused a strong response when administered alone at a particular point in time or concentration tended to inhibit ERK activation in response to a physiologic estrogen. But at other times or concentrations, the same xenoestrogen might cause a weak response on its own, in which case it would tend to enhance ERK phosphorylation in response to physiologic estrogens.

There were exceptions to these general patterns, however, which highlights the need to study effects of individual xenoestrogens at different points in time, at varying concentrations, and in different tissues. The effect of shifts in the patterns of ERK activation are only just beginning to be explored, although it is known that these patterns constitute an important component of information flow within a cell. The correct flow of information is likely to be especially critical during windows of vulnerability that are based in part on life stage.

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